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Original Articles.

SUB-ACUTE COMBINED SCLEROSIS OF THE SPINAL CORD,[1]

AND ITS RELATION TO ANEMIA AND TOXEMIA.

By Charles L. Dana, M.D.

There is a group of cases that may be characterized broadly as forms of ataxic paralysis of subacute course. They are not so very uncommon, and it seems to me they should be better known as representing a distinct clinical type. The trouble is essentially a spinal one. It occurs chiefly during or just after middle life, and is associated with a cachexia, sometimes with pernicious anemia, and with other toxemic states. Attention was called to this condition first by Dr. J. J. Putnam, and then by myself seven years ago (*Journal Nerv. and Mental Dis.*, Feb., 1891, April 1891). A little later Dr. Grainger Stewart (*British Med. Jour.*, 1891,) reported a case evidently belonging to the group, quoting our papers. At least ten articles recording similar cases have been published since then, not including those specially belonging to pernicious anemia. The important papers were those of Leyden[2], Rothmann[3], Teichmuller[4], Boediker and Juliusburger[5], Bowman[6], Nonne[7], Rhein[8], Mitchell and Rhein[9]. The whole subject was carefully monographed in 1895 by Rothmann and in 1896 by Bastianelli[10]. None of these writers, however, except the last named seem to have seen the articles by Putnam and myself. Most of them, in studying this subject, have been intent on the anatomical findings, and have tried chiefly to establish a specific organic change in the columns of the spinal cord. Their collections of cases have been confusing on account of the variety of the clinical pictures associated with the combined sclerosis.

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[1] Read before the New York Neurological Society, Nov. 1st, 1898. For discussion on this paper see page 37.
[2] Zeitschrift für klinische Medicin, Bd. 21, H. 1-2.
[3] Deutsche Zeitschrift für Nervenheilkunde, 1895, VII.
[4] Deutsche Zeitschrift für Nervenheilkunde, 1895, VIII

It is my object especially to call attention to the fact that there is a definite disorder recognizable by its symptoms and course, as well as by the anatomical changes found after death. Dr. Putnam quite recently told me that he had seen a good many cases of the type since his original article. I have seen six cases in the last three years, and the disease is apparently more common than multiple sclerosis, and nearly as common as the spinal atrophies.

Much direct and side light has been thrown upon the nature and symptoms of the malady by a number of investigators who have studied the pathological changes of the spinal cord in pernicious anemia.[11]

They found that in pernicious anemia there was sometimes, but not always, a pathological change in the spinal cord.

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[5] Neur. Centralblatt, 1896, p. 326.

[6] Brain, Summer, 1894.

[7] Arch. f. Psych., XXV., 1893. Deut. Zeitschrift f. Nervenheilkunde, VI.

[8] Rhein, Jour. Nerv. and Ment. Dis., Nov., 1896.

[9] Mitchell and Rhein, Jour. Amer. Med. Assoc., April, 1898.

[10] Bull. d. R. Acad. Med. di Roma, 1895-6.

[11] In 1887, Lichtheim, Verhandlungen d. VI. Congress für innere Medicin, Wiesbaden, 1887, reported a series of cases of pernicious anemia, in which he described changes in the spinal cord. His investigations were followed by those of Eisenlohr, Deutsche med. Wochenschrift, 1892; Von Noorden, "Untersuchungen über schwere Anämien," Charité-Annalen, 1891-'92; Minnich, Zeitschrift für klinische Medicin, vol. XXI, Ma. 1-2, vol. 22; Nonne, Archiv für Psychiatrie, vol. XXV; Burr, University Medical Magazine, 1895; Lloyd, Journal of Nervous and Mental Disease, 1896; James Taylor, Medico-Chirurgical Transactions, vol. LXXVIII; and Riggs, International Medical Magazine, September, 1896.

This consisted of a degeneration which always affected the posterior columns, and in most cases both the posterior and lateral columns, but never the lateral columns alone. The gray matter and the nerve roots were rarely touched, and the change was of the nature of a degeneration of the nerve tracts without any later shrinking of the cord, as in locomotor ataxia. The blood vessels sometimes showed hyaline changes, but were not always much diseased, though small hemorrhages were now and then seen. These changes of the spinal cord peculiar to pernicious anemia were, as will be seen later, not strictly like those seen in the class of cases to which I now refer.

With this brief summary of previous work I proceed to relate the history of my own case:

Summary.—M., age 50. No syphilis, no lead, no alcoholism. Spring, 1897, numbness in right hand, then in left, ataxia and weakness. Later, girdle sensation, weakness in limbs, with numbness, emaciation. Progressive weakness, paraplegia, some anesthesia of lower and upper extremities, pains in back and legs, loss of control of sphincters, no severe anemia, death in one and a half years.

Autopsy.—Spinal cord, degeneration throughout whole length of posterior and lateral columns, confined mostly to columns of Goll and crossed pyramidal tracts. Recent acute softening in anterior horns and columns at lower cervical and first dorsal segments, same in lower dorsal segments. Anterior horns affected at some levels. Some disease of blood vessels, small hemorrhages.

History.—Converse L., native of Maine, 50 years of age, single; by occupation, an engineer. His father died of stricture of the esophagus, one brother died of some mental trouble following typhoid fever, and a sister had consumption. There is no history of any definite nervous disorder in the family. The patient had always been a healthy man, of good habits. He drank moderately. There was no history of syphilis.

In 1861 he had typhoid fever, and in 1875 he suffered from an attack of round worms. In 1886 a heavy pipe fell on his head and stunned him for about ten minutes. In other respects his previous history was negative.

About one year before he was seen by myself, and without any known cause he noticed that the fingers of the left hand became numb. This numbness soon extended to the right hand, but to a less degree. The numbness was, in the course of a few weeks, associated with some awkwardness in the use of the hands, and also with some actual weakness in the arms.

He was able to work, however, until about two months before admission. He then felt a sensation of a band around the waist, and developed a weakness in the lower extremities, which was also accompanied with some numbness and with some wasting of the muscles. This anesthesia and weakness of the arms and legs continued until he became unable to walk, and he was admitted to Bellevue Hospital on Jan. 3d, 1898. At that time he could stand, but not walk; he could use his hands with difficulty on account of the ataxia rather than weakness. He complained of pain in the arms, neck and back, and of sensations of coldness and of muscular twitchings in the legs. He suffered from insomnia. An examination of the lungs, heart, abdominal viscera and urine showed no morbid changes. The urine was acid, clear, its sp. gr. was 1.028; it contained a trace of albumin, but no sugar. There was a general emaciation, and he said that he had lost about thirty pounds in the last few months. He was anemic, but not strikingly so.

Examination.—There was a paresis of the lower extremities, especially of the right. The extensors and flexors of the right leg were almost entirely useless, and those of the left were very weak. The muscles of the right thigh were almost entirely paralyzed, so that he could with difficulty draw up or extend, abduct or adduct, the thigh. This paralysis involved more especially the right thigh. There was weakness of the arms, but he could move them in all directions, the right arm being much the worse. His co-ordination, however, was very bad. The grasp of the dynamometer in the right hand was 45, in the left 60. The circumference of the forearms was only 6 inches, and that of the calves only 10¾ inches. The knee-jerks were present and exaggerated. The superficial reflexes were also exaggerated. There was a diminution of the galvanic reaction of the muscles of the legs and arms, but no degenerative reaction.

In the feet and legs tactile sensation was blunted and delayed, muscular sense lost and temperature sense inaccurate. In the arms tactile sense was delayed and blunted, but not to a great extent. Temperature, pain and muscular sense, however, were very much disordered, and the loss of muscular sense, with consequent ataxia, was most marked of all. His pupils reacted to light and accommodation, no nystagmus was present, visual acuity and fields of vision were normal, hearing was slightly impaired, taste was blunted, smell was normal. He had no disturbance of speech or swallowing. His temperature was normal; his pulse ranged from 72 to 90, respiration from 18 to 20.

The general symptoms, therefore, were those of a patient suffering from a somewhat spastic and ataxic paralysis of the legs, with marked ataxia and some weakness of the upper extremities.

The anesthesia involved the upper extremities, reaching to near the shoulders, and the legs, reaching to the lumbar region, but not the trunk. The tactile anesthesia was slight, the temperature and pain anesthesia greater, the muscular anesthesia most marked of all, especially in the arms. The earliest and most marked characteristic sensory disturbance was the numbness and ataxia in the arms. This was followed by tactile anesthesia, and then by a moderate amount of muscular weakness in the arms.

The next set of symptoms were those involving the legs and feet; the sensory symptoms appeared first, followed quite rapidly by weakness of the legs with stiffness, exaggeration and then lessening of the reflexes, and finally by a nearly total paralysis.

The patient looked emaciated and very slightly anemic. He had, however, no striking blanching of the skin or mucous membranes, not enough, at least, to attract attention or lead to an examination of the blood. He had no hemorrhages at any time, and no special disturbances of the stomach or intestines. His mind was clear until the last week of his illness, and he showed no signs then of any loss of memory or dementia. He had no convulsive seizures of any kind. His paralysis was not accompanied with any tremors or any fibrillary twitchings. He had considerable pain in the back and limbs.

The patient continued under observation until Feb. 8, a period of over six weeks. During this time the paralysis of the legs became greater until it was nearly complete. The anesthesia increased slightly. He lost control over the bladder. The condition of the arms did not change so much, but there was a steady decrease of power. He suffered during this time from pains in the legs and back, but the mind continued clear, and there was no involvement of the special senses, other than that mentioned.

He was given iodide of potassium, arsenic and iron, and was treated symptomatically for his pains and insomnia with codeine and trional. He gradually became weaker; on Feb. 7th his temperature began to rise, and he became entirely unconscious. On Feb. 8th the temperature rose to 102 deg., pulse to 108, and he died from exhaustion.

Autopsy.—The autopsy was made by Dr. C. Phillips, of the Carnegie Laboratory.

The specimens of the brain and cord were placed in alcohol and Müller's fluid, and sections were made and stained with hematoxylin, carmine and by Nissl's and Weigert's methods.

The results of the autopsical examination outside of the nervous system showed nothing of great importance, the changes being mostly such as would be found after great emaciation. The muscular system was fully developed, the skin pale and unelastic and with but little subcutaneous fat. The lungs showed no tuberculosis, but were adherent to the chest wall. The heart was small, the left ventricle contracted, weight 9 oz. Some atheroma of the coronary arteries was found. In connection with the subject of pernicious anemia, it is important to note that the stomach was very small, having a capacity of only about 300 cubic centimeters, and measuring only 16 centimeters in length. The mucous membrane was normal. The small intestines and mesenteric glands were normal. The spleen was enlarged and the kidneys also were enlarged, but showed no other change than a congestion. The suprarenal bodies were normal. The liver weighed 2,095 grams, but showed nothing abnormal, though Quincke's test was not tried.

The skull cap was symmetrical and showed rather deep but regular markings. The brain membranes were normal. The brain itself was of average size and the convolutions symmetrical and normal in appearance. There was some increase in the intraventricular fluid. The blood vessels at the base of the brain showed a few areas of arterial sclerosis. These were limited to the circle of Willis. The brain otherwise appeared normal.

The Spinal Cord.—On opening the spinal column the dura mater appeared quite pink in color, slightly thickened and partially adherent over the cervical and lumbar portions. Its blood vessels were normal. The pia mater appeared to be markedly congested in the area reaching from the fifth to the eighth cervical and from the fifth to seventh dorsal regions; also in the lower lumbar. In the sacral regions were a few small areas of subpial hemorrhage. The substance of the cord seemed firm, except in the region of the eighth dorsal segment, where it tended to assume a flattened appearance. In the first, third and fifth sacral segments were small grayish-white masses which were differentiated from the balance of the cord. They varied in size from one to two centimeters in diameter and were firm in consistency.

Microscopical examination showed a brown atrophy of the heart muscle, a chronic diffuse nephritis, and a simple atrophy of the liver with pigmentation.

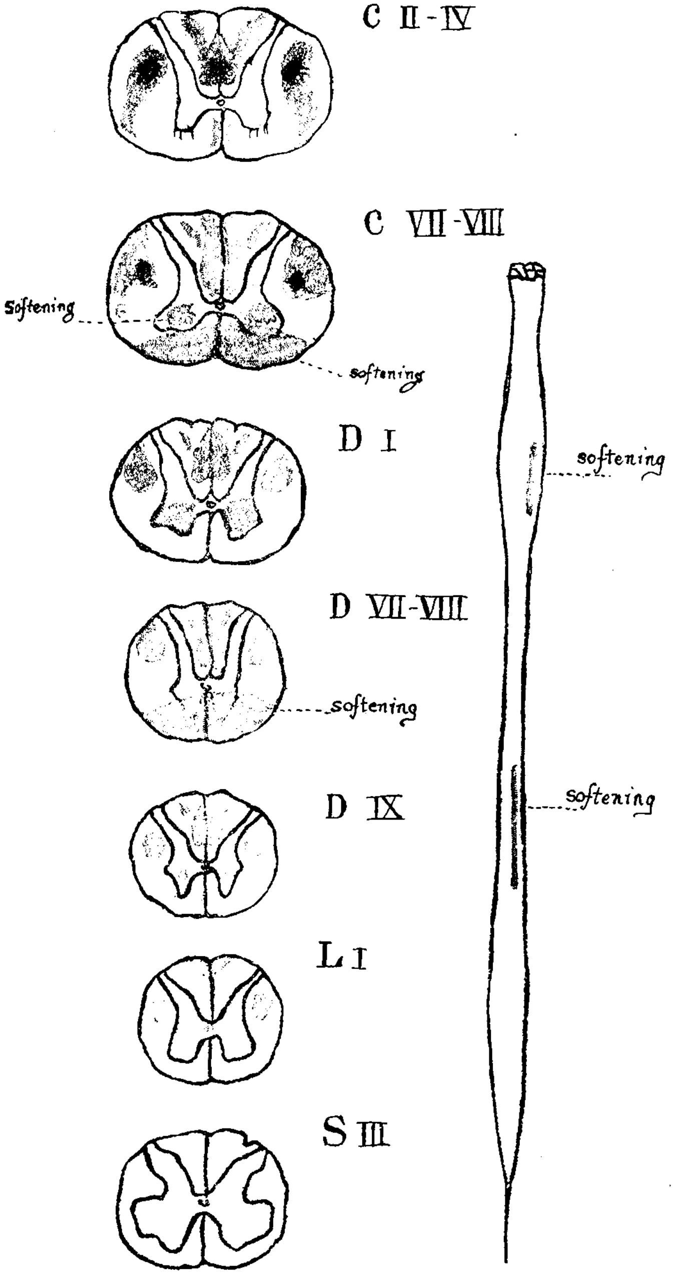

FIG. I. Showing the foci of softening and the location of the systemic degeneration.

MICROSCOPICAL EXAMINATION OF CORD.

Sections of the cord were made at the level of the 2, 4, 6, 7, 8, cervical; 1, 4, 7, 8, 9, dorsal; 1 lumbar and 3 sacral segments. They were stained by Dr. Phillips, to whose help I am much indebted.

Second Cervical Level.—Pia mater practically normal. Anterior columns; (Fig. II) slight degenerative change in the left anterior median column. Lateral columns, marked vacuolar degeneration in the region of the pyramidal tracts, extending to the periphery. Very few nerve fibers can be seen here; only a wide-meshed reticulum of connective tissue; a degeneration of the antero-lateral ascending tracts. Direct cerebellar tracts not affected. The lateral limiting layer shows occasionally a few vacuolar spaces. Posterior columns show a degeneration involving the deeper parts, including the columns of Goll, but leaving comparatively untouched the V-shaped area near the periphery. The root zones are not affected. The gray matter of the anterior horns is moderately healthy, but some change can be seen in the nerve cells (Fig. II). The anterior and posterior roots show some loss of nerve fibers, but no extensive change. The blood vessels of both the gray and white matter show thickened walls, with what I take to be a *hyaline change* in them. There is, undoubtedly, an increased vascularity of the whole section of the cord; in some places the blood vessels are dilated and in others collapsed, and here and there are a few minute extravasations, but there is *no evidence of true inflammation.*

Fourth Cervical.—(Fig. III) The changes here resemble those just described, but are much more marked. The vascular change appears more active, and there is more involvement of the anterior horns. The oldest process at this level seems to be in the deep parts of the posterior columns.

Sixth Cervical.—(Fig. IV) No decided change can be made out in the anterior median or anterior fundamental columns. In the lateral columns the change is confined chiefly to the pyramidal tracts, spreading thence apparently to the periphery and involving the direct cerebellar tracts. The process is most serious and oldest in the posterior columns which have undergone throughout nearly their whole extent a degenerative change. The anterior horns are also at this level partly destroyed by softening, leaving empty areas. Where the gray matter is left one can still find good anterior horn cells. The vascular changes here are also rather active. One sees long vessels reaching from the meninges directly to the anterior horns, where they apparently end in dilatations, and sometimes in punctate hemorrhages. Some of the vessels have thick

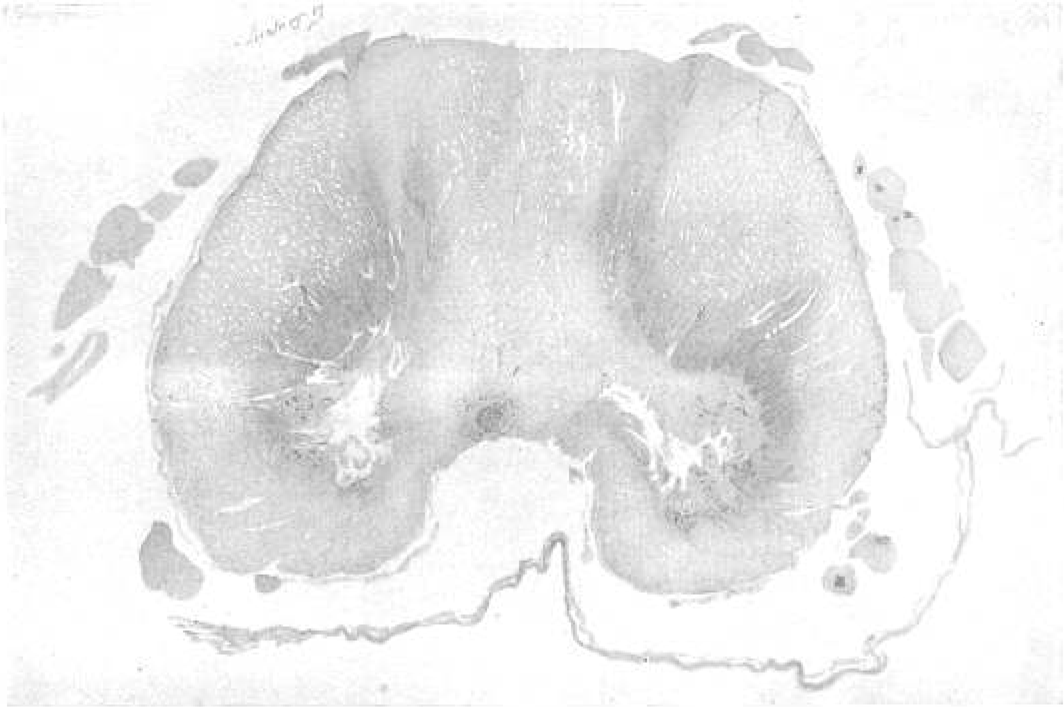

Fig. IV. Sixth cervical segment. The anterior horns are partly softened, leaving a cavity.

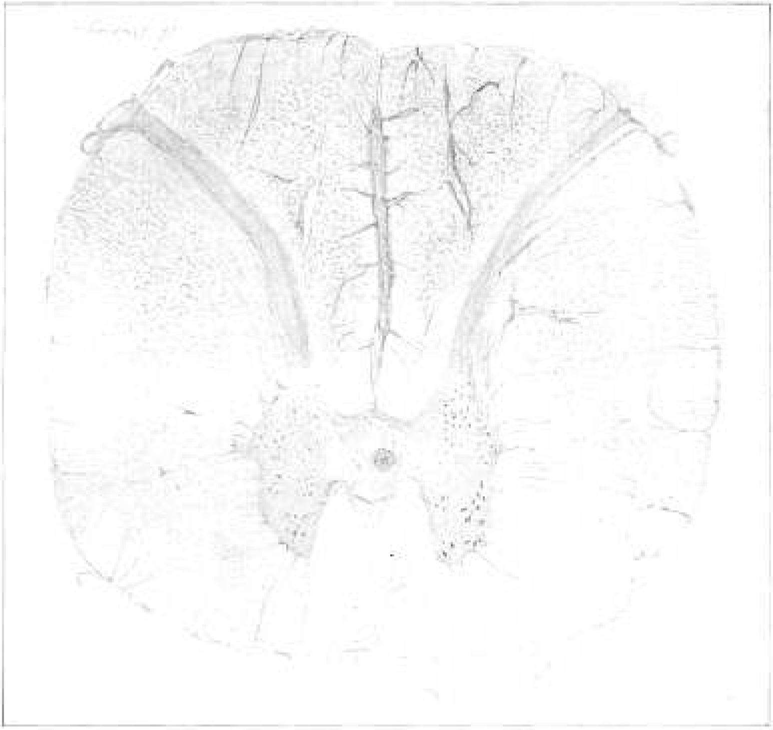

FIG. II. Second cervical segment. The degenerated areas are indicated by the vacuolated appearance of the section.

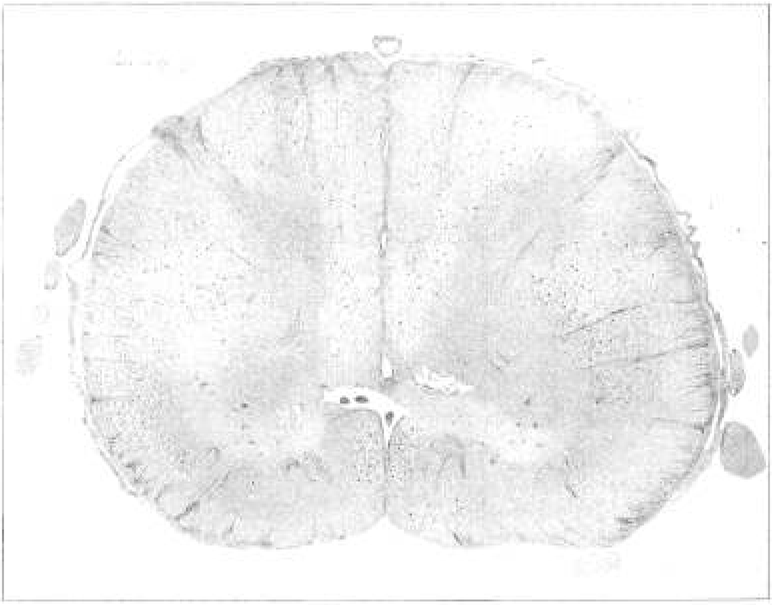

FIG. III. Fourth cervical segment. (The reproduction does not show very perfectly the intensity of the process.)

walls and look collapsed. The picture suggests the possibility, therefore, of a *softening* in part at least, from *thrombosis.*

Seventh and Eighth Cervical.—Here a large part of the anterior columns and part of the lateral columns have been destroyed or broken away. The loss of these parts, as shown in the figure (Fig. V), is probably due, however, to a difficulty in manipulating the softened cord at this point, and not due to the fact that there was an actual primary softening in this region. Weigert's stain at this level shows that the degeneration, though very marked and striking, is limited in the lateral columns almost entirely to the crossed pyramidal tracts, extending slightly into the lateral limiting layer. The direct cerebellar tracts and Gowers' tracts are not affected. The posterior columns are very extensively involved, but areas of normal fibers are found here in the posterior fundamental column and in the root zone on each side. The blood vessels here also show thickened walls and appearances similar to the sections higher up. The *anterior roots* show degeneration of fibers.

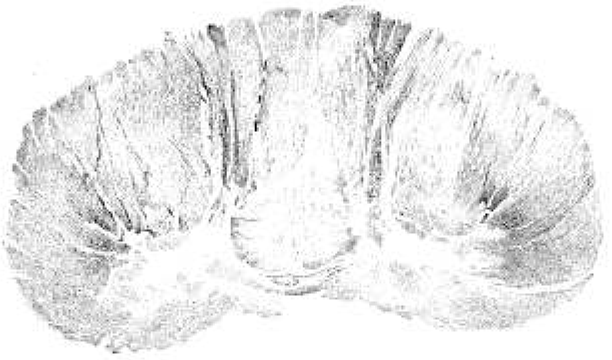

FIG. V. Eighth cervical segment.

First Dorsal.—Here the change is similar to that in the lower cervical, but less marked. The anterior horns are so badly softened that they have dropped out, leaving open spaces. The degeneration in the lateral columns is still mainly confined to the crossed pyramidal tracts. Here the sclerosis is dense in its central part, and one sees numerous dilated and tortuous blood vessels, but no hemorrhages, and no proliferations or exudations. The direct cerebellar tracts and Gowers' columns are not affected. The area of greatest activity of the pathological process is still deep in the posterior columns.

Fourth Dorsal.—This shows again a sclerosis affecting the lateral and posterior columns as much as in the first dorsal, but with less intensity. The anterior horns are also less involved. The cells of the columns of Clarke are somewhat affected, but apparently only secondarily. In the anterior horns there is a great deal of active vascular change and several *small hemorrhages* (Fig. VI).

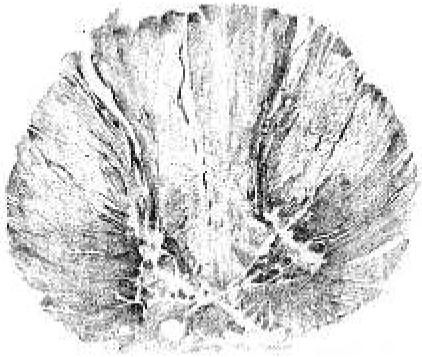

FIG. VI. Fourth dorsal segment.

Sixth to Seventh Dorsal.—The anterior horns here, in fact, the anterior third of the cord, have fallen in as a result of softening and post-mortem manipulation. (Fig. VII) The degenerative process in the remainder of the column shows more activity than was present in the segment of the dorsal cord

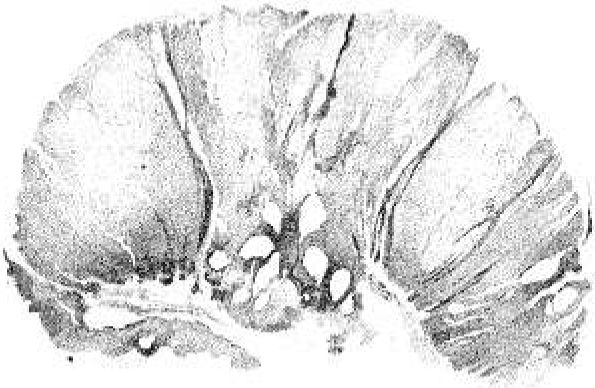

FIG. VII. Sixth to seventh dorsal segment.

above. The change is more extensive in the lateral columns, but even here it does not reach the periphery, and does not involve the cerebellar or Gowers' tracts. But the softening is more profound and the vacuolar spaces more extensive. Similar evidence of the more active and extensive degeneration is present in the posterior columns. The cells of Clarke's columns are normal on one side, but have been destroyed on the other. Widely dilated vessels and small hemorrhages can be seen in the anterior horns, parts of which have softened and dropped out. The anterior horn cells, however, which remain are in fair condition. The anterior roots show in their centers large dilated vessels, but no very extensive degeneration. Neither here nor above has there yet appeared *any sign of meningitis.* The vessels do not seem to have as much thickening of the walls as in the cervical region. Examples of the degenerating anterior horn cells (Nissl stain) are shown in Fig. VIII.

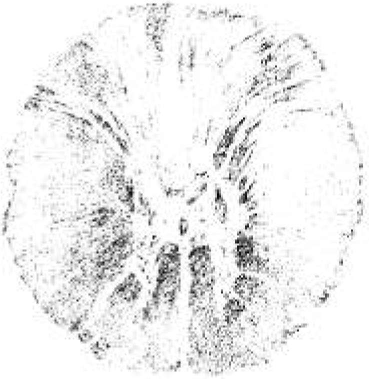

FIG. IX. Ninth dorsal segment.

Ninth Dorsal.—Here the degeneration is most marked in the posterior column, especially the posterior and median. It is present in the lateral columns, but moderate in intensity, reaching, however, to the periphery, and to some extent perhaps involving the direct cerebellar tracts, but not the tracts of Gowers. Lissauer's columns are normal. Small hemorrhages can sometimes be seen in the sclerotic parts here, and other sections show the presence of numerous amyloid bodies (Fig. IX).

First Lumbar.—Here there is a slight sclerosis confined to the crossed pyramidal tracts, and a very slight change also in the peripheral portion of the posterior columns in their median part. The anterior horn cells here are normal. The third sacral segment is practically normal (Fig. X).

To summarize the foregoing we find that there were two original foci of disease, one in the lower cervical zone and one in the lower dorsal region, the upper being the older. The earliest part attacked was the posterior columns, especially in the central and deep parts. The process extended to the posterior external columns at some levels, but never entirely involved the root zones. The lateral columns were badly diseased also, especially at the two levels mentioned. The crossed pyramidal tracts were the most affected, but the disease extended at the cervical level to the direct cerebellar tracts and Gowers' columns. Below, the disease was limited to the crossed pyramidal tracts so sharply that it might be called secondary, but the microscopical appearances were almost too active and acute for this. The change in the pyramidal tracts was much less noticeable high up in the cervical region, and could not have been secondary to any cerebral or bulbar process, though other parts were not examined microscopically. They were macroscopically normal. The gray matter of the anterior horns was seriously diseased, and in parts entirely softened. (Figs. XI and XII.)

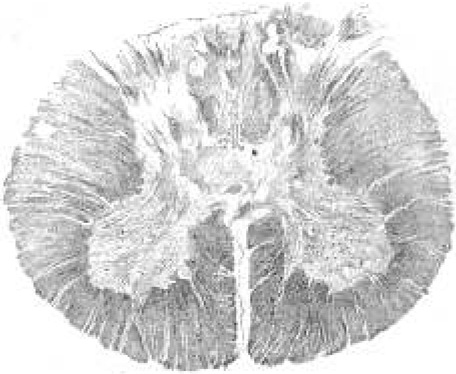

FIG. X. First lumbar segment.

Shortly before death there was a central softening, involving the anterior horns and part of the anterior columns at the level of the lower cervical and mid-dorsal segments. It was just such a process as might be produced by a thrombosis of the anterior median artery. Microscopical examination showed this process to be a degenerative change, in which the nerve fibers were in great part destroyed, leaving large vacuoles (vacuolar degeneration). This was followed in the older areas by proliferation of connective tissue, and a true sclerotic change. In some levels there were small hemorrhages into the anterior horns. The blood vessels, in parts, were certainly diseased. This vascular disease involved the median and outer coats, especially the former, and seemed like a hyaline thickening. It was not like the peripheral periarteritis of syphilis. (Fig. XIII.)

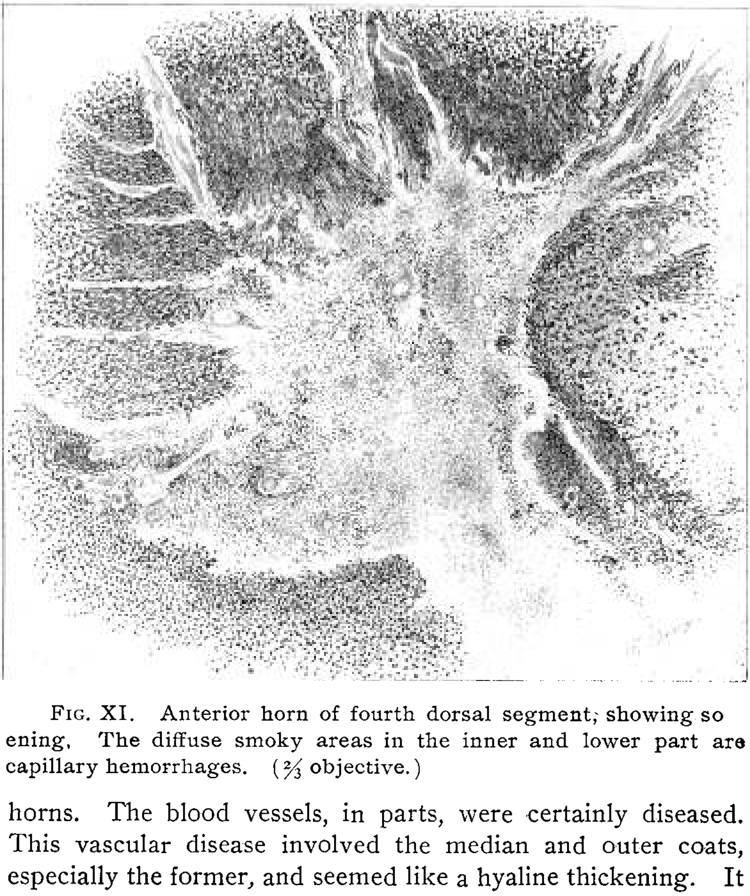

FIG. XI. Anterior horn of fourth dorsal segment; showing softening. The diffuse smoky areas in the inner and lower part are capillary hemorrhages. (⅔ objective.)

REVIEW OF CASES AND HISTORY OF THE DISEASE.

Several collections of cases of combined sclerosis have been made in the last twelve years, and attempts naturally followed to make out the description of diseases due to such scleroses, yet not belonging to paresis, tabes, hereditary ataxia, chronic myelitis or disseminated sclerosis[12]. I tried this myself eleven years ago, but came finally to the conclusion that nothing very definite could be recognized, and that most, if not all of the cases called by Gowers "ataxic paraplegia," were aberrant or complicated forms of tabes, transverse myelitis or multiple sclerosis.

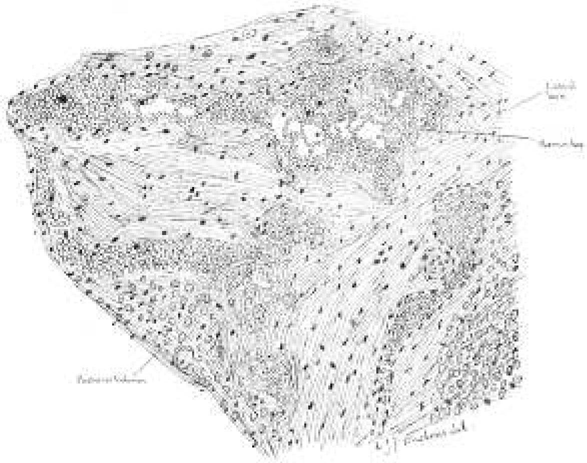

FIG. XII. Showing capillary hemorrhages in the anterior horn of the fourth dorsal segment. (1/6 objective.)

The time has come, however, when this view need not be taken. There seems to be without doubt a disease which has features of its own, both clinical and anatomical, and which need not be confounded with those above mentioned. I have analyzed seventeen such cases reported with autopsies. This, perhaps, does not include all, but I have taken those which seemed to me to represent the type. I have excluded, for example, a case of Rothmann occurring in a syphilitic person and two of Boediker and Juliusberger occurring in the insane. Rothmann tabulates twenty-seven cases, but he includes, in my opinion, irregular, complicated or doubtful forms. I find that but one sure case among Nonne's collection. Eight cases were reported by American neurologists (loc. cit.).

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[12] Omerod, Brain, April, 1880; Grasset, Archives de Neur., 1886, Nos. 32, 34; Dana, Medical Record, July 2d, 1897; Rothmann, Deut. Zeitsch. f. Nervenh., 1896.

Etiology.—The disease occurs oftener in men, according to statistics, but my clinical experience is against this. Four out of my six cases were women. The age of most cases is between 50 and 60 (9 out of 17), and next to this between 40 and 50 (5). The youngest case was 36 (Rothmann's). There is often some family or personal neuropathic history.

The disease follows acute infections like influenza, prolonged diarrheal or dysenteric attacks, possibly shock, lead poisoning, malaria, lathyrism (?) and pellagra (?). One patient had a malignant tumor. Syphilis is not a cause. The relationship to profound secondary and to pernicious anemia is very important. In about 10 per cent. of cases, and perhaps more, pernicious anemia undoubtedly exists; in others there is usually a very marked anemic and cachectic state. Often, if not always, however, nervous symptoms develop before evidences of extreme or extended anemia are present.

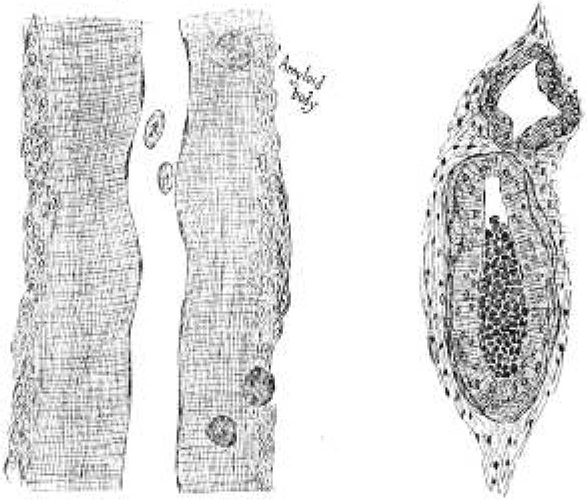

FIG. XIII. Showing cross-section and longitudinal section of small arteries. The specially thickened middle coat should be represented as quite homogeneous.

Symptoms.—The initial nervous symptom is nearly always a persistent paresthesia usually of the feet, associated with some weakness of the part. Following this there is a good deal of ataxia, and this symptom increases with the loss of motor power. Tactile, thermic and pain anesthesia are not marked until late in the disease, but the patient often has severe pains in the back and limbs. There may be some differentiation of sensibility as in syringomyelia. With the weakness of the legs there is at first an increase of knee-jerks, and sometimes ankle clonus and rigidity, so that the patient shows the symptoms of spastic and ataxic paraplegia. Later the spasticity may become less and the knee-jerks disappear.

The arms are involved after a few months, the symptoms beginning with paresthesia, weakness, awkwardness and a very slight anesthesia. The disease may begin in the arms, as in my second case. The mind may be slightly weakened, the memory becoming defective and the patient emotional. The cranial nerves are not often involved, though optic atrophy has been once noted. Limitation of visual field occurred in one case seen by me with Dr. Markoe. The blood presents evidences of anemia. Usually this is secondary, but in a considerable number of cases the typical findings of pernicious anemia are present. The following records are taken from a typical case of combined sclerosis; the patient, a woman of about 60, having eventually pernicious anemia. The first examination was made in the early stage (first 6 months) of the disease; the last when the patient had become nearly paraplegic (1 year). The examinations were made by Dr. Frederic E. Soudern at the request of Dr. Frank H. Markoe, who had charge of the patient, and to whom I am indebted for permission to use the records.

The disease progresses rather rapidly, though remissions occur. In from six months to a year the process has nearly reached its height. During this time the patient emaciates, marked anemia or pernicious anemia sets in. The skin is sallow and pale, and pigmentation is seen. Diarrhea occurs at times. The bladder becomes weak and retention of urine follows; the control of the rectum is eventually lost, and the patient lies bed-ridden with paralysis and contractures of the lower limbs. The arms do not become so badly paralyzed. Death occurs in from one-half to two years in the fatal cases. It may be prolonged to three years, and it is not unlikely that some patients can be relieved or the process checked if seen in time.

Pathology.—I have already spoken at length on this point. The disease is a toxic one, but whether due to the failure of some glandular tissue of the body to act or to infection or poison is not known. As the disease belongs to the degenerative period of life, it may fairly be supposed that in some cases the blood-making organs undergo premature senility, and that the nerve centers are poisoned in consequence. In other cases less easily demonstrable metabolic perversions occur. It is not likely that the change in the spinal cord is primary.

BLOOD EXAMINATION OF A TYPICAL CASE OF COMBINED SCLEROSIS.

	January, 1898.	October 1st, 1898.	October 27th, 1898.
Number of red cells per 1 c.m.m. (Thoma)	3,000,000	2,100,000	1,840,000
Number of white cells	5,640	3,400	6,000
Differential Count—			
Small lymphocytes	30 per cent.	34 percent.	38.5 per cent.
Large lymphocytes	9 per cent.	6 per cent.	13 per cent.
Polymorpho-nuclear neutrophiles	46 per cent.	53 per cent.	44.5 per cent.
Eosinophiles	3 per cent.	2 per cent.	2.5 per cent.
Abnormal Forms—			
Myelocytes	12 per cent.	5 per cent.	1.5 per cent.
Amount of hemoglobin (Fleischl)	85 per cent.	61 per cent.	52 per cent.
Specific gravity	1,057	1,050	1,047
Color index	1.4	1.5	1.4
Fibrin	Diminished	....	....
Blood plates	Diminished	Red cells large, of fair color; many poikilocytes, macrocytes, and microcytes; few normoblasts and some megaloblasts.	Red cells abnormally large and of good color; otherwise same as October 1st.

As to the location of the trouble, the posterior columns of the spinal cord are the parts first and most involved. The process attacks these columns in their entire length, but especially involves the postero-median parts. It appears to start in certain foci in the lower dorsal or cervical cord, and to spread thence. New foci apparently develop as the disease progresses; the lateral columns are also severely affected, and especially the crossed pyramidal tracts. The other columns are involved, but less regularly and completely. The lower dorsal and lower cervical levels of the cord are especially selected. The anterior horns are attacked and very severely, but the change here comes later. Finally, there may be actual softenings in the cord, producing cavities. The blood vessels are somewhat affected with hyaline degeneration, but I am unable to say how important a factor this is. The walls are thickened, there are in places dilatations and hemorrhages, and in other places collapsed vessels. There is no evidence of inflammatory reaction. The meninges are not involved, nor are the anterior and posterior roots much affected. Examinations of the peripheral nerves (Rothmann) and of the brain have been negative.

Diagnosis.—The disease is easily distinguished from locomotor ataxia by the absence of syphilitic history, the rapid onset, the anemia, the motor weakness, the absence of ocular symptoms and of lightning pains. There is usually also at first an exaggeration of the reflexes. The steady and rapid progress of the malady is also most characteristic.

Multiple neuritis is excluded by the slow onset, the marked ataxia, the absence of muscular wasting, tenderness and pain, and the development of bladder and rectal symptoms.

I must confess that my last case presented such a marked differentiation of cutaneous sensations that I believed it to be one of syringomyelia. The necropsy showed a cavity in the cervical cord due to softening, and in a measure explained, if it did not justify, the diagnosis. The steady and rapid progress of the symptoms ought to exclude such a diagnosis.

There is little in common with the ataxic paraplegia of Gowers in this disease. This latter trouble in my opinion, can always be sifted down to some form of tabes, chronic myelitis or disseminated sclerosis. Still, some cases of tabes of irregular type suggest at first this malady, and it may require some time to enable one to make the distinction.

The existence of marked anemia, and especially of pernicious anemia, is, taken with ataxia, paralysis and rapid course, pathognomonic.

The essential nature of this process is a primary nerve degeneration, affecting apparently the nerve tissue first. The same poison which causes pernicious anemia does the work here, only it may affect the nervous centers without causing a true essential anemia. It seems even as if in some cases the neural change came first.

I can only say in conclusion that much light can in the future be thrown on this trouble by a study of milder clinical cases, for such, no doubt, occur. We may find it related to some definite infection which does not always hit the nerve centers with killing force. It may even be that the disseminated sclerosis of early life is a malady of the same kind, but one which does not progress on account of the youthfulness of the tissues. The tendency of neuropathology now is to lessen rather than increase the number of pathogenic processes of the nervous system, and it may well be that this combined sclerosis is only a familiar poison attacking the predisposed nervous centers of those in the declining years of life.

MENTAL DISEASES AMONG THE ARABS. Milhon. (Annales Medico-Psychologique, 8th Series, Vols. III. and XIV., 1896-1897.

From observations made upon the Arabs in Algiers, Milhon presents some statistics regarding their sanity. The Arabs, he claims, are as a rule, a race with sound and healthy minds, mental diseases among them being rare; and the chances for such, as compared with the French, though his statistics are made from a very small number of cases, are as 1 to 134. Alcohol in "Kiff" and syphilis seem to be the causes of such cases as are found; and as the excessive use of alcohol is becoming more common, the tendency will probably increase. The effects of the intoxication caused by Kiff are obscenity, sexual excitement, laughing, ecstacy, hallucinations of sight, and sensations as of flying. The author suggests that the religious views of the people, their tendency toward fatalism and mysticism are the causes of their more common forms of hallucination which are generally of the exalted type.

JELLIFFE.